right, uvula to the left. Trigeminal nerve normal in every particular. Taste diminished somewhat over anterior of the tongue.

Hearing diminished during the activity of the zoster, later became augmented in the right ear. Electrical examinations in the early days showed increased faradic and galvanic irritability. Later the faradic became gradually diminished, muscle and nerve, and at present very strong currents elicit faint responses. Galvanic shows no polar change, though the contraction is slow and vermicular.

The case was especially of interest, on account of the moderate amount of pain, which in geniculate lesions is usually severe and persistent.

Dr. J. Ramsay Hunt said that the case reported by Dr. Abrahamson was quite typical, presenting the characteristic syndrome of the herpetic inflammation (posterior poliomyelitis) of the geniculate ganglion, *i. e.*, an eruption of the herpes zoster within the zoster zone for the geniculate ganglion (herpes oticus), a peripheral facial palsy, and hypoacusis. He had personally examined the case, and thought that the auditory disturbance was due to the inflammatory swelling of the tissues of the external auditory canal and tympanic membrane, and was not dependent upon a central involvement of the acoustic nerve or its terminations. Symptoms of Ménière's disease, not infrequently observed in this group of cases, were entirely absent. He emphasized the red and swollen appearance of the auricle, which was displaced laterally from the side of the head, and the narrowing of the external meatus, which no doubt, in not a few cases, gives rise to errors in diagnosis, such as perichondritis and diffuse inflammation of the auricle. The single large bleb referred to on the tympanum had been observed in other cases; within the canal and tympanum the vesicles often show a distinct tendency to coalesce.

Dr. Hunt desired to place on record a case that he had seen a few days previously which represented a different type of the same group of cases. This was a case of herpes occipito-collaris with seventh nerve palsy occurring in a man 71 years of age who developed a typical herpes zoster in the left side of the neck and occipital region. Eight days later there appeared a typical facial paralysis on the same side which had been preceded by tinnitus the day before. Taste perception was diminished on the anterior two thirds of the tongue on the affected side, and there was a slight but distinct diminution of hearing of neural origin on the same side. The facial palsy involved all branches, but very slight movement was still present in the upper and middle branches. There was no evidence of any eruption within the zoster zone for the geniculate ganglion. The speaker explains a case of this type on the basis of an inflammatory reaction in the geniculate ganglion.

MYOTONIA ATROPHICA.

By D. J. Ramsay Hunt, M.D.

Myotonia atrophica is an extremely rare affection; and so far as Dr. Hunt was aware these are the first cases of this affection recorded in this country. In a recent study of this subject by Pelz, the percentage of myotonia cases showing atrophy is given as 11 per cent. In the following two cases, myotonia and muscular atrophy are present in two brothers. Another brother, now dead, is said to have presented somewhat similar symptoms:

Case 1. The first patient was a man, 44 years old, a native of Germany. His occupation was that of cleaner in a barroom. He stated that he first noticed a weakness in his hands about ten years ago, and six years later in the lower extremities. Three years ago the subjective symptoms of myotonia first manifested themselves, *i e.,* while the first movements of the arms and legs were somewhat stiff, this would diminish and finally disappear after repeating the movements several times. The weakness and associated atrophy had gradually grown worse. He had had no pains nor paresthesia.

At examination the patient was well built and of good muscular development. His face was dull and lacked expression. His speech was slow and somewhat labored and monotonous. He walked with a steppage gait due to bilateral foot-drop, and on lifting the arms a wrist-drop became apparent. The muscles of the trunk, the shoulder and hip girdles and the thighs were unusually well developed. In the upper arms, the forearms and the calf muscles a well marked tapering atrophy was present, which was especially marked in the triceps, the ulnar side of the forearm and the extensor surfaces of the calves. The muscles supplied by the cranial nerves showed no defect, save the dulness of expression and the articulation disturbance. There were no pupillary changes. The tendon reflexes of the arms and lower extremities were abolished. The skin reflexes were present. The myotonia on voluntary movements was only distinctly demonstrable in the hands. On making a fist, the hands and fingers slowly relaxed in the typical manner, the stiffness growing much less after a series of the same movements. While the subjective disturbances were present to a slight degree in the lower extremities, they were not objectively demonstrable. No myotonic disturbance on voluntary movement was present in the eyelids, the jaws, neck, trunk, upper arms or lower extremities. The mechanical irritability of the muscles was increased and showed typical myotonic characteristics (persistence of the contraction) in the muscles of the face, tongue, arms, shoulders, thighs and calf muscles. It was exquisitely present in the intrinsic muscles of the hands. Gross motor power was well preserved in all the muscles of the body excepting those of the arms and legs. Flexion and extension of the elbow were very much diminished in force. There was almost complete paralysis of the extensors of the wrist and fingers; the flexors were also very weak, as were the intrinsic muscles of the hands. Flexion and extension of the knee were well preserved on both sides. There was, however, almost complete paralysis of the peroneal and extensor muscles of the foot. Plantar flexion of the ankle and toes, while present, was considerably diminished. There were no objective disturbances of sensation. The extensors of the wrist and fingers and the peroneal and extensor group of the anterior tibial region failed to respond to strong faradic or galvanic currents. The posterior muscles of the calves, the intrinsic muscles of the hands, the flexors of the wrist and fingers and the deltoids showed the myotonic reactions.

Case 2. This patient was a brother to the first case shown by Dr. Hunt. He was a man, 41 years old, a baker, whose symptoms dated back eighteen years and began with a weakness in the hands and arms. He found that he could not lift heavy objects, that his hands would not close tightly, and he stated that a boy could open his fist. He was positive that the weakness preceded the stiffness by many years, the myotonic rigidity first manifesting itself ten years ago. The trouble in the lower extremities dated back about ten years, and consisted of weakness and stiffness. Before that the patient could run and jump and was a good dancer. At that time he found that upon arising from a sitting posture he had to wait a brief period before a certain stiffness wore off. He had also had some difficulty with speech; his tongue seemed heavy and he had difficulty in getting started in talking, but after talking a while, this slowness would wear off. A few years ago this disturbance of articulation was worse than it was now. He ate rather slowly and chewed his food for a long time; otherwise he had no difficulty in mastication or deglutition.

Status: The patient was well built and of strong muscular development. He bore a marked resemblance to his brother (Case 1), not only in physique, but also in face and coloring. His gait was slow and shuffling, with distinct steppage character (foot-drop). The facial expression was dull and heavy; articulation was slow, monotonous and indistinct. The muscles of the trunk, neck, shoulder girdle, hip girdle and thighs were unusually well developed and showed no signs of weakness or of atrophy. The upper arms, forearms and lower legs were distinctly tapering and atrophic, especially the lower portion of the triceps, the ulnar side of the forearm and the anterior muscle group of the lower leg. Flexion and extension of the elbow were weak, the supinator longus muscle was practically absent on both sides. Extension of the wrist and fingers was so very much diminished that it could be easily overcome by the pressure of a single finger. The flexor movements of the hand and fingers were also very feeble. In the lower extremities the dorsal flexion of the feet and toes was practically abolished. Plantar flexion, while present, was very much reduced. The pupils and cranial nerves were negative; the arm jerks and the Achilles jerks were absent; the knee jerks were present but very much diminished, reinforcement being necessary. All the skin reflexes were present (plantar flexion of the toes). The mechanical irritability of the muscles was increased and showed the characteristic response in the intrinsic muscles of the hands, the forearms and upper arms, deltoids, calf muscles, feet, face and tongue. The only demonstrable myotonic disturbance on voluntary movement was in the hands; here the typical rigidity persisted after a contraction, and gradually relaxed as the movement was repeated. Subjectively, the symptom of myotonia was also present in the face muscles of articulation, and in the lower extremities. The myotonic electrical reactions were demonstrable in the muscles of the lower face, the tongue, the deltoid, the upper arm and hands, and in the posterior muscles of the calves. The muscles of the forearm, especially the extensor group, showed a very markedly diminished response to both currents. The electrical reactions were practically absent in the anterior tibial and peroneal group of the lower extremities.

Dr. B. Sachs said the first case shown by Dr. Hunt bore a close resemblance to Thomsen's disease, but on account of the associated atrophies and the appearance of the arms it reminded him of the Charcot-Marie type or leg type of progressive muscular atrophy. The condition of the forearms, and of the legs, suggested that type.

Dr. L. Pierce Clark said that in a case reported by Kaiser, in 1897, there were three conditions present, namely, hypertrophy, atrophy and myotonia; the conditions apparently followed a cerebral palsy. The hypertrophy occurred in those muscles that were paralyzed.

Dr. Dana said he had examined these patients at his clinic, and the idea had occurred to him that they might more properly be called cases of muscular dystrophy with myotonia. The dystrophy came on before the myotonia. They were perhaps examples of one of the forms of progressive dystrophy of the Charcot-Marie type upon which the myotonia had been implanted.

Dr. B. Onuf also agreed with Dr. Dana that the cases probably represented a myotonia implanted on a primary muscular dystrophy. The flaccidity of the muscles in one of the cases shown was extremely marked. In the other case the facial expression was so typically mask-like that it presented a very striking feature, and rather spoke against its belonging to the Charcot-Marie type.

Dr. Dana, in reply to Dr. Onuf, said that Gowers had reported one case of progressive dystrophy of the Charcot-Marie type in which the mask-like expression of the face was present.

Dr. Hunt, in closing, said that those who had written most extensively on this subject had tried to determine if this represented a distinct clinical type of atrophy, and whether it was a spinal or myopathic type, and the consensus of opinion was that it could not be brought under one of the well-known groups of atrophies. The atrophies in the cases he had shown seemed to be confined chiefly to the arms and forearms, with preservation of the muscles of the hands.

RESEARCHES ON THE EPILEPTIC VOICE.

By L. Pierce Clark, M.D., and E. W. Scripture, M.D.

Dr. Clark stated that for several years he had held that there was a characteristic voice in epilepsy which, if accurately analyzed, would possibly be an aid in the differential diagnosis, and with the help of Dr. Scripture's methods of voice study, which had been explained and illustrated at the April, 1907, meeting of the Society, he was now able to give a demonstration and analysis of the epileptic voice type. It was unnecessary to say that there was no past literature to refer to in such studies, as both the problem and results were entirely novel. After explaining and illustrating in detail the method of obtaining these records, Dr. Clark showed that in the voice curves from epileptics there was a peculiarity which struck the eye at once. Instead of the continual rise and fall in melody of the normal voice, vowels and phrases ran along on even tones. The speech of an epileptic was, in fact, "plateau speech"; his melody proceeded by even tones, There was also a tendency to monotony, and the successive sounds did not vary much from each other as normal ones did. This defect of speech of the epileptic could be easily removed by training, but relapses occurred quickly. The degree of permanency of the removal was still to be proven. The speaker said he had seen the defect remain for several years after the epileptic attacks had ceased. He had no adequate explanation to offer for this voice defect in the epileptic. There was no parallel; no similar defect in any patient suffering from any other motor disorder of the nervous system, insanity or imbecility. Two of the record cases he had shown were entirely free from the bromides. They had universally found this epileptic type of voice in all well-established cases of epilepsy of whatever type. As a tentative explanation, they offered the theory that the voice was due to the chronic exhaustion of the nerve centers peculiar to epilepsy. The diagnostic value of the voice sign was obvious.

Dr. Charles E. Atwood said he had had the opportunity of following